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The Effect of Exertional Hypertension Evoked by Weight Lifting on Vascular Endothelial Function

To the Editor: Chronic hypertension is associated with arterial endothelial dysfunction, the precursor to atherosclerosis (1); however, less is known about the effects of brief episodes of hypertension on endothelial function. Lamping and Dole (2) demonstrated that <5 min of moderate elevations in perfusion pressure reduces conduit coronary arterial endothelial function for up to 2.5 h. This endothelial dysfunction also extends to the human microcirculation (3).

Cardiovascular benefits of exercise extend to both aerobic and resistance exercise and include improved endothelial function (4). However, weight lifting elevates systolic blood pressure (SBP) up to 400 mm Hg (5), possibly impairing endothelial function. We examined whether brief elevations in arterial pressure during weight lifting impair brachial arterial endothelial function assessed by flow-mediated vasodilation (FMD).

Nine men and 5 women, age 28 ± 2 years (mean ± SE), who engaged in regular weight training (1 h, 3 times per week) constituted the conditioned weight lifter (CWL) group. Eleven men and 5 women, age 33 ± 2 years, who did not regularly engage in exercise constituted the non-weight lifter (NWL) group. Those with known risk factors for coronary endothelial dysfunction or with prior adverse reaction to nitroglycerin were excluded. The Institutional Review Board of the Medical College of Wisconsin and of the local General Clinical Research Center approved the protocol.

Brachial artery FMD was assessed before and within 1 h after leg press exercise. Ultrasound imaging of brachial artery diameter and flow velocity was performed using standard methods (6) at baseline and after a 4.5-min suprasystolic blood pressure cuff inflation. This process was repeated after 30 min of resistance exercise, followed by assessment of endothelium-independent vasodilation using 0.4 mg sublingual nitroglycerin (NTG).

Diastolic brachial artery diameters were digitally recorded from longitudinal images and analyzed using an automated edge-detection algorithm (Medical Imaging, Iowa City, Iowa). Percentage dilation was calculated from baseline to peak values obtained after cuff release (FMD) or NTG.

Subjects performed 2 to 3 sets of 6 to 8 repetitions each to near maximal exertion, serving as their own controls. Brachial artery diameter, FMD before and after weight lifting, peak SBP during exercise versus baseline were each compared using a paired *t* test. Changes in FMD (%) between groups were compared using an unpaired *t* test. Correlations between FMD before and after exercise were made using Pearson correlates. Data are reported as mean ± SE with $p < 0.05$ considered to be significant.

The mean age of all subjects was 31 ± 1 (range 21 to 40) years. There were no differences in arterial blood pressure, heart rate, or lipid profiles between CWL and NWL groups, and these values were within the normal range (data not shown). However, 1 CWL and 1 NWL subject had previously undetected hypercholesterolemia (LDL 181 and 170 mg/dl, respectively) but normal FMD and were included in analysis. One CWL subject and 3 NWL subjects had systolic hypertension.

The CWL and NWL subjects increased SBP during weight lifting (Table 1). Basal brachial artery diameters were similar between CWL and NWL before and after weight lifting. The FMD and dilation to NTG were unchanged by exercise in CWL (Fig. 1). However, FMD was reduced in NWL after exercise ($p = 0.001$ vs. baseline and vs. CWL). Dilation to NTG and peak hyperemic flow velocity in NWL and CWL were similar (Table 1). There was no correlation in either group between the change in FMD and cholesterol, baseline BP, or maximal weight lifted (data not shown).

The primary findings of this study are that: 1) endothelium-mediated vasodilation is impaired by acute exposure to exercise-induced hypertension in healthy sedentary subjects; and 2) no impairment exists in individuals who perform regular resistance exercise. These data suggest that acute resistance exercise associated with hypertension impairs endothelial function in unconditioned subjects and that chronic resistance training protects against this vascular dysfunction.

The Health Professionals' Study demonstrated an inverse correlation between weight training and cardiac events (4). However, vascular disease occurs in high-performance athletes, possibly related to elevations of BP during training (7). We observed vascular dysfunction after brief hypertension during heavy lifting in sedentary subjects.

Based on animal studies (2), the magnitude of hypertension generated in the present study should impair endothelial function. However, no reduction in function was observed in trained athletes. There are several possible explanations. Exercise increases cardiac output and therefore shear stress, stimulating nitric oxide (NO) production (8). Alternatively, vascular antioxidant capacity may be improved by chronic endurance exercise training (9). Shear stress up-regulates superoxide dismutase expression and activity in vascular endothelium (10). Finally NO-independent mechanisms of dilation (e.g., endothelium-derived hyperpolarizing factor) resistant to acute hypertension (11) may prevail in CWL subjects.

Noninvasive measures of BP during weight training underestimate direct intra-arterial measurements; therefore, our estimations of stimulus intensity may be low (12). Induced hypertension in the present study was more transient than in most studies (3) but is sufficient to induce impaired endothelial function (2). Twenty to 60 min lapsed between the end of exercise and the second vascular study. Endothelial dysfunction may have occurred in regular weight lifters but was too transient to demonstrate on repeat testing. This is unlikely, however, because less intense hypertension impairs endothelial function for hours (2). Some subjects were taking dietary supplements that may have independent effects on

Table 1. Hemodynamic and Vascular Responses to Resistance Exercise

	Weight Lifters	Non-Weight Lifters
Maximum weight lifted (lbs)*	439 ± 58	213 ± 29
Maximum SBP (mm Hg)*	213 ± 8	196 ± 4
Increase in SBP during exercise (mm Hg)	95 ± 9	78 ± 5
Resting HR after exercise (beats/min)	68 ± 4	61 ± 3
Baseline artery diameter (mm)	4.3 ± 0.2	4.0 ± 0.2
Post-exercise artery diameter (mm)	4.2 ± 0.2	4.0 ± 0.2
Change in FMD after exercise (%)*	1.4 ± 1.1	−4.7 ± 1.1
Pre-exercise peak change in flow velocity (%)	71 ± 8	65 ± 10
Post-exercise peak change in flow velocity (%)	83 ± 12	64 ± 8

Note: All results are expressed as mean ± SEM. *Significant difference between weight lifters and non-weight lifters ($p < 0.001$).

FMD = flow-mediated vasodilation; HR = heart rate; SBP = systolic blood pressure.

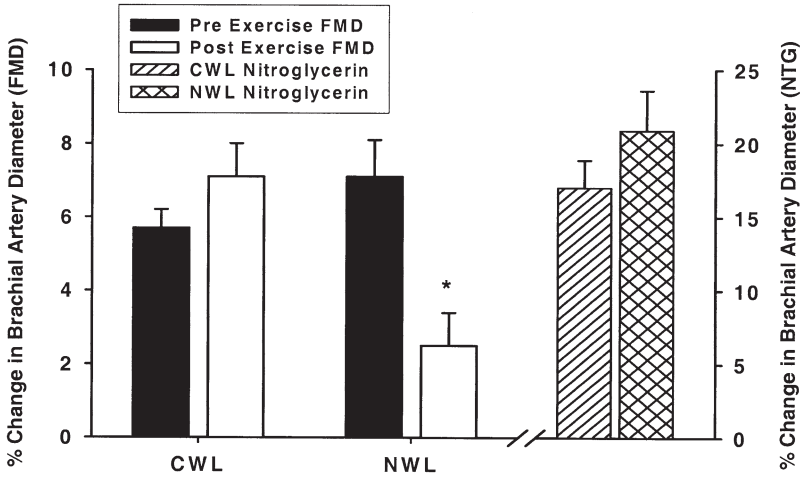

Figure 1. Effect of exercise-induced hypertension induced by resistance exercise on endothelium dependent flow-mediated vasodilation (FMD) in conditioned weight-lifters (CWL) and nonconditioned weight-lifters (NWL). There was no difference in the brachial artery responses to flow before and after exercise in CWL (5.6 ± 0.5% at baseline to 7.1 ± 0.9% after exercise; p = NS), while there was a significant reduction in FMD after compared to before resistance exercise in NWL (7.1 ± 1.0% at baseline to 2.5 ± 0.9% after weight lifting; *p < 0.05 compared to pre-exercise value NWL). There was no difference in the brachial artery responses to endothelium-independent nitroglycerin (NTG; 0.4 mg) between groups (20 ± 2.6% vs. 17 ± 1.9%; p = NS).

endothelial function. Finally we cannot exclude a direct endothelial effect of exercise, independent of SBP in NWL. However, the fact that we measured FMD in a nonexercised extremity suggests that a direct effect of exercise, unless due to circulating factors, is not likely.

In summary, lower-extremity exercise acutely impairs brachial endothelial function in sedentary but not conditioned weight-lifting subjects. This reduction in vascular function may increase the propensity for atherosclerosis and vascular events. Understanding how chronic exercise protects against vascular dysfunction may have important implications for new therapeutic strategies aimed at primary prevention.

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